

A Twenty-Year Experience with Surgery for Ureteral Reflux*

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It is a real honor to be asked to return to Richmond, particularly for a Northern boy, to give the first R. Carl Bunts Lecture. Many in the audience will wonder why all this fuss about the retirement of the head of a small department in a VA Hospital in Richmond. Those who wonder only do so because they did not have the privilege of knowing Bunts well. I could say that Bunts was a great leader, a great scientist, and a great physician. I could say that Bunts had no children and that he and his lovely wife, Dori, have made Bunts' residents their lives and family. While all these things are true, they fall woefully short of the mark in describing or attempting to explain why there is all this fuss about Carl Bunts. Bunts is being honored because he is a rare human being, the kind of person that most of us meet only once or twice in a lifetime. I had the honor of being Bunts' fourth resident from 1948 to 1951. He cast his spell over me then, and as I have met the other residents through the '50's and through the '60's and now into the '70's, I sense in talking to them that he has done the same thing to them now that he did to me and to my contemporaries 25 years ago. Maybe it is magic, maybe Bunts has congenital charisma, maybe Bunts has a quality that our young people strive to achieve, namely, being a warm human being.

With this introduction, I will move on to the scientific part of the presentation which deals with 22 years of experience in vesicoureteral reflux, most of which, of course, started right here in Bunts' department.

When I began my residency in Richmond, Vir-

ginia in 1948, urologists were aware of vesicoureteral reflux as an entity. It was known to occur in patients with badly distorted urinary tracts, particularly those with obstruction at or below the bladder neck, and those with urinary tract tuberculosis or neurogenic bladders. No one sought out reflux *per se*, and if it was found, it was not considered to be causative but rather the result of the distorted urinary tract. Certainly, no one thought of treating reflux *per se* but rather in treating the disease that was causing the reflux. This apathy towards reflux is reflected in the fact that during the five-year period from 1943 to 1948 no articles on reflux appeared in the *Journal of Urology*. Since our hospital in Richmond was a paraplegic center, we were more aware of reflux than most urologic departments throughout the country. We were doing two-film cystograms on all of our paraplegic patients when I arrived there in June, 1948, and we had been doing this for some years. We felt great concern about the progressive dilatation of the upper urinary tract that we were observing in some paraplegic patients. The theory was that these urinary tracts were dilating because of an obstruction at the ureterovesical junction, and our therapy was aimed at cutting out this obstructive segment and reimplanting the ureter into the bladder in such a way that the obstruction could not occur. We used the same technique in paraplegics that we used in patients with bladder tumors in whom we did a partial cystectomy with ureteral reimplantation into the bladder. We kept groping for a better method of performing ureteral reimplantation. We used the fish-mouth technique; we also tried the direct mucosal-to-mucosal type of anastomosis that Cordonnier was then popularizing in ureteral sigmoid anastomosis. We made some abortive attempts at tunnel formation by passing the ureters through the bladder wall obliquely, but it must be remembered that we were operating to overcome obstruction and that

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our main effort was to secure the ureter in its new location in an unobstructed manner. The results of these operations were disappointing. Improvement was rarely demonstrated either clinically or by x-ray. Early in my residency, we performed five consecutive operations based on these principles. In three patients, all function was lost from the involved kidney, and in two, function was preserved only by nephrostomy drainage. We now know that these operations failed because they did not stop the reflux, but this was not appreciated then. At this time, the prevailing feeling among urologists was that operation for megaloureter at the ureterovesical junction was contraindicated because the results were often so disastrous.

Dr. Bunts suggested that I attempt to find out what was happening to the urinary tracts of patients with neurogenic bladders with the passage of time. We had a wealth of material to draw from, including the records and x-rays of several hundred paraplegics taken since the founding of the department in 1945. These x-rays were mostly IVP's, some retrogrades, and a large number of cystograms. Reviewing this material case by case, I became aware that some of the paraplegic patients were developing a specialized type of saccule or diverticulum that was located at the point where the ureter was entering the bladder. Certainly these bladders contained many saccules and diverticula, but there was something different in the appearance about this particular saccule and its consistent location. I set aside a number of cystograms demonstrating this saccule and showed them to Dr. Bunts. After reviewing these x-rays, we arranged to cystoscope these patients to determine whether there was any connection between the saccule and the ureteral orifice. Cystoscopy proved that the saccule was always above and lateral to the trigone; the ureter always ran down the floor of the saccule. In each case the catheter passed through this segment with no obstruction, and there was no thickening of the intravesical ureter to account for obstruction of any type. From these observations it was apparent that the ureteral hiatus was dilating. In the process of dilation, the intravesical ureter was falling into the saccule that resulted and was losing the support of the bladder muscle behind it. It seemed that the logical method to correct this was to excise the saccule surgically and to push the ureter into the bladder and then to close the bladder behind the ureter.

We knew that we had to repair the bladder

muscle behind the saccule, but we did not know what to do with the mucosa. At that time, I felt that was not very important. We decided to sew the mucosa under the ureter. Unfortunately, this proved to be a mistake and was the technical defect that caused some of the early operations to obstruct. The simple logic involved was appealing, and since our efforts to correct the dilated upper urinary tract of the paraplegic had produced such poor results, Dr. Bunts was more than willing to try this new operation. Therefore, on May 17, 1950, with Dr. Bunts as my assistant, we did a left ureterovesicoplasty on a 28-year-old paraplegic patient with left reflux. We believe that this was the first time any person was ever operated on specifically to correct reflux. To our joy and amazement, three-week and six-week postoperative cystograms showed no reflux, and the postoperative intravenous pyelogram looked better than the preoperative one. Encouraged by our success, we performed our second operation on June 23, 1950. It, too, was successful. Our first bilateral ureteral vesicoplasty was done on August 31, 1950. By April 1951, we had operated on 9 patients and 11 ureters and had successfully stopped the reflux in 8 of the ureters and had encountered no obstruction.

Dr. Austin Dodson, our consultant, had been watching our series develop, and he suggested that it be presented to the prize essay contest sponsored by the American Urological Association, rather than submitting it to the *Journal of Urology* through regular channels. This essay was awarded first prize and was presented before the American Urologic Association in Chicago in June 1951. This resulted in the elimination of Dr. Bunts' name as co-author since he was not eligible under the rules of the essay contest. If the paper had been submitted to the *Journal of Urology* through regular channels, Dr. Bunts would have been a co-author, and the operation would have been the Hutch-Bunts operation from the first. While this did not seem particularly important at the time, it resulted in the fact that many urologists do not appreciate the vital role played by Dr. Bunts in the development of the original ureteral vesicoplasty.

Dr. Ruben Flocks then asked me to spend a year at my alma mater, the University of Iowa, and to try the operation on some of his patients with meningomyeloceles and primary reflux. It was at Iowa City that I became aware of the tremendous difference in reflux in children with primary reflux

and in paraplegics. The children with primary reflux had none of the gross changes in the bladder wall that were so characteristic of the paraplegic patients, nor could I find saccules at the ureterovesical junction. This was distressing because my explanation of the etiology of reflux in paraplegics was that the changes in the bladder wall created a sacculum at the ureterovesical junction which damaged the valve mechanism resulting in reflux. This theory could be extended to include reflux in patients with true infravesical obstruction such as prostatic hypertrophy, urethral strictures or urethral valves because these patients, like the paraplegic, had vesical trabeculation and sacculum formation, but it could not possibly explain the most common type of reflux (primary reflux). Cystoscopically, these children had normal appearing bladders. The only positive cystoscopic finding was golf-hole orifices which we could not explain satisfactorily at that time. The results of our surgical experience in Iowa City were published in the *Journal of Urology* by Dr. Raymond G. Bunge, Dr. Ruben Flocks, and me and reported the first series of antireflux surgery in nonparaplegics. This series contained several brilliant successes, but our enthusiasm was dampened somewhat by the appearance of the most feared complication: obstruction at the operative site. In looking back on this series, we got our good results in the patients with primary reflux and our failures in the meningo-myelocele group. During the next five years antireflux surgery gained some advocates. Eugene St. Martin and his group published reports of a favorable nature. Dr. Bunts, on the East Coast, and I, on the West Coast, continued to use the operation, but its acceptance was impeded by fear of obstruction at the operative site. Dr. Bunts and I felt a personal responsibility for this operation and polled the members of the American Urologic Association who had used the operation to determine the results they had obtained. During this period, many of the operative failures were sent to me for a second operation. Actually, this proved to be a blessing in disguise, because it was in re-operating on the patients that I realized that the basic defect in the original operation was sewing the mucosa under the transplant.

The first modification of the original operation came in 1958. This was by Dr. Wyland Leadbetter and Dr. Victor Politano. It included all the features of the original operation and did so without a suture line under the transplant. It also handled the mucosa

correctly by passing the ureter through a submucosal tunnel. The second operation or modification was presented by Dr. Al Paquin. Here the ureter was cut off outside the bladder and brought through a new opening high up in the bladder wall and down through a submucosal tunnel. The Paquin operation originally incorporated a Vest nipple to prevent stenosis of the cut end of the ureter. This operation was highly successful and is widely used today with a number of modifications. I believe at the time of Dr. Paquin's untimely death, he felt that the nipple was no longer necessary.

It is my belief that when we successfully stop the reflux, we stop all further attacks of acute pyelonephritis, and we make the patients clinically well. On the other hand, the pyelonephritic changes on the IVP preoperatively never change; they will remain forever. Any hydroureter or hydronephrosis present preoperatively, however, will slowly return to normal. When we stop the reflux, we stop any further progression of the renal deterioration. We are converting active pyelonephritis into healed pyelonephritis. No function loss prior to surgery can ever be regained. Successful antireflux surgery does not assure that the bacteriuria will not recur. I believe that the source of the bacteriuria is the urethra flora and that when the reflux is present those bacteria have immediate access to the kidney. Following successful antireflux surgery, the bacteria still have easy access to the bladder urine, but if there is no longer any reflux the infection will be limited to the bladder.

To evaluate antireflux surgery fairly, we must discuss three different time periods. The first time period runs from 1950 to 1958. The second period runs from 1958 to 1968, and the third period runs from 1968 to the present. The reported results in the 1950 to 1958 period, when the *Hutch I* was the only operation available, were about 80% completely satisfactory (defining completely satisfactory as stopping the reflux without causing obstruction at the operative site). During the second period, due to the efforts of many urologists working to improve the operation, 90% were completely successful. During the last five years over 95% were completely successful. Fortunately, from the first, antireflux surgery has been evaluated against solid x-ray criteria; by this I mean preoperative IVP's and cystograms and postoperative IVP's and cystograms. This has given a validity to the reported results in the antireflux surgery not present in many types of surgery.

During the last 15 years, we have lived through what I like to call the cystogram explosion. During my residency we rarely did cystograms on anyone. Then all of a sudden we did many cystograms, and we learned a lot about the role of reflux in all types of urinary disease. If we do an IVP we get a lot of information, but the IVP alone is not enough to be certain that a serious urological disease is present. We also get a lot of information from the cystogram, but the cystogram alone is not enough to give urologic clearance. I have the feeling that if we have the combination of a normal IVP and a normal cystogram in a patient with urinary tract infection that this patient has almost no chance of going on to uremia from pyelonephritis. If we could screen everyone in the United States with urinary tract infections with an IVP and cystogram, we could eliminate those patients in whom both studies were negative. Almost all of the patients in whom the possibility of progressive pyelonephritis exists will have a positive cystogram or a positive IVP, or both, on the initial screening examination. All of us have been giving urologic clearance on the basis of a negative pyelogram and a negative cystogram, and it is very rare that a patient whose first work-up shows a negative cystogram and negative pyelogram will ever get into serious trouble subsequently. These patients may have more attacks of urinary tract infections, but they will be limited to the bladder.

Any patient with a proven urinary tract infection should be worked up and classified as in Table 1. A careful history should be obtained, and if the urinary tract infections are occurring without fever, the history is classified as cystitis. If the infections are occurring in the presence of fever, the history is classified as pyelonephritis. The cystogram is read as showing or not showing reflux, and the pyelogram is read as showing or not showing the changes of pyelonephritis. A patient classified in this manner falls into one of five groups. The overwhelming ma-

jority will be in the cystitic group. They will have a cystitis history, a negative cystogram, and a negative pyelogram. A smaller group of patients will have a history of pyelonephritis, yet their x-ray work-up is negative. This is classified as Grade I pyelonephritis. Patients with Grade II pyelonephritis have a positive cystogram, yet the pyelogram is still normal. In Grade III pyelonephritis, the cystogram shows no reflux, but the pyelogram shows pyelonephritis. In Grade IV pyelonephritis, both the cystogram and the pyelogram are positive.

Once your patient has been properly classified, a logical plan of management is easy. The patient who has cystitis and Grade I pyelonephritis has in common a negative pyelogram and negative cystogram. This is the group of annoying but basically harmless urinary tract infections. This type of patient can be given intermittent antibacterial therapy with confidence that the patient does not have a serious urological disease. Patients with Grade II pyelonephritis may be treated with constant antibacterial therapy or with antireflux surgery. Patients with Grade III pyelonephritis have a positive IVP but no reflux. Most of them are adults, and most of them got their pyelonephritis because they had reflux when they were children. I have pointed out that changes of pyelonephritis stay in the kidneys forever. Fortunately, most of the Grade III pyelonephritis represents a healed pyelonephritis and no treatment is warranted. However, there are some sleepers in this group. These are patients in whom the initial cystogram shows no reflux, not because the reflux has actually disappeared but because it has become intermittent. When this possibility is suspected you must repeat the cystogram until the reflux is found. The classification then changes to those patients with Grade IV pyelonephritis. Patients with Grade IV pyelonephritis have a pyelonephritic history, pyelonephritis on the pyelogram, and reflux on the cystogram. I believe that all of these patients should have antireflux surgery.

TABLE 1

CLASSIFICATION	HISTORY	REFLUX	PYELONEPHRITIC CHANGES
Cystitis	Cystitis	No	No
Pyelonephritis			
Grade I	Pyelonephritis	No	No
Grade II	Pyelonephritis	Yes	No
Grade III	Pyelonephritis	No	Yes
Grade IV	Pyelonephritis	Yes	Yes

PANEL DISCUSSION

Dr. Paul Langlois: Dr. Hutch, you did not mention the adult female with symptoms only of cystitis, without chills or fever, without high back pain, flank pain and so forth, who does have reflux.

Dr. Hutch: This would be a patient with a cystitic history who has reflux on the work-up. We studied patients who had a cystitic history and found normal pyelograms and cystograms in all but seven. It is

very rare to have a cystitic history and a positive finding on either the cystogram or the pyelogram. When this does happen, the patient should be graded not according to the history but according to the x-ray findings, so that your patient in spite of her cystitic history should be Grade II pyelonephritis.

Dr. Joseph Fiveash: The child with the golf-hole ureter is no problem in management nor is the child with the wisp of reflux who has no changes in the upper tracts. Do you still believe in the maturation of the intravesical ureter, and if so, do you have any hints to tell us which child will mature the ureter and which will not?

Dr. Hutch: We have all had many patients who outgrow their reflux due to some process of maturation. We all have our own criteria. In patients with Grade II pyelonephritis where we have normal kidneys and proven reflux, we have two courses—1) constant antibacterial therapy or 2) antireflux surgery. We

all develop our own methods to determine which ones we are going to follow. We have to individualize these cases and each patient is so different.

Questioner: I would like to ask you if you go along with Lyons urethral ring?

Dr. Hutch: Yes, I think that it is the urogenital diaphragm.

Questioner: Are there cystoscopy changes in patients with primary reflux.

Dr. Hutch: There is no question that in primary reflux there are two things that are actual facts; one is that the trigones are very large. This means that the orifice is abnormally lateral, and they also have golf-hole orifices. Thus, any theory that is going to explain primary reflux has to explain the golf-hole, and it also has to explain the megatrigrone and the gradations that Dr. Lyons and Dr. Tanagho talk about which are excellent in bringing this information into an organized form.